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RESPIRATION IN AN ATMOSPHERE UNDER HIGH PRESSURE[1]

G. V. Troshikhin[2]

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ABSTRACT: The electrical activity of the external intercostal muscle and the time of the respiratory cycle were investigated in adult Wistar rats during exposure to various gas mixtures at increased pressure.

Increased density of inspired gas mixture, due either to an increase in external respiration or physical characteristics of the inert gas, used for dilution of the oxygen, produces an increase in the amplitude of the electrical pulses in the respiratory volleys and the time of the respiratory cycle. A reaction of this kind in a dense medium is the consequence of an increase in the resistance to respiration and a rise in the load on the respiratory musculature. The paper also considers the mechanism of the increase in resistance to respiration in a pressurized atmosphere.

/1808

Exposure of man to an atmosphere under high pressure has a significant effect on the organism. This problem has become particularly acute for physicians and physiologists concerned with diving in conjunction with the current exploration of the ocean shelf. Increasing the pressure leads to an increase in the density of the ambient gas, affecting primarily the respiratory function. In a pressurized atmosphere, man displays a slowing of respiration and a decrease in the maximum pulmonary ventilation [11, 15, 19, 23, 24], as well as a slight increase in the vital pulmonary capacity, noted by several researchers [6, 22]. It is natural to assume that the principal role in the development of these changes is played by the high density of the inspired gas. Thus, it has been found that the breathing of a dense gas at atmospheric pressure (a mixture of

[1]Paper delivered at the Symposium entitled "Function of External Respiration in an Altered Gas Medium," Leningrad, 22 January 1971.

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*Numbers in the margin indicate pagination in the foreign text.

oxygen and sulfur hexafluoride) produces essentially similar changes in external respiration [14, 15].

Since the pressure differential between the alveolar space and the ambient medium which allows the passage of gases through the respiratory pathways, is produced by the activity of the respiratory musculature, breathing a dense gas increases the load on the respiratory muscles [7, 22]. Increasing the resistance to respiration and imposing a considerable load on the respiratory musculature are the principal factors which limit prolonged stays of man under high pressure [13].

It should be pointed out that there are comparatively few studies devoted to the influence of increased density of inspired gas on the respiratory function; we have almost no data on the electrophysiological analysis of the activities of the respiratory musculature of the organism when in a pressurized atmosphere.

Therefore, we have attempted in this paper to use electrophysiological /1809
means to determine the load on the respiratory muscles in experimental animals (rats) as a function of the level of the excess pressure and the density of the gas mixture.

Method

For this purpose, we built an apparatus in the laboratory which consisted of a cage-stand for the animal, an electric stopwatch with calibrations for precise recording of the time of the respiratory cycle as well as amplifying and counting devices for studying the electrical activity of the respiratory musculature. The cage-stands were in the form of narrow oval tubes constructed of plastic, with movable side walls. In a cage of this type, the rats can remain in a resting state for several hours without being held down. Carbon sensors were firmly fastened to the sidewalls of the cage and connected to an electrical circuit with a step-by-step selector. The latter made it possible to record on an electronic stopwatch the time of each ten respiratory cycles and to record the electromyogram for the same period. The biopotentials were recorded by means of two silver electrodes, 0.2 mm in diameter, implanted in

the outer intercostal muscle in the vicinity of the fifth and sixth intercostal spaces. The electrical activity of the muscles was transmitted through a UBP 1-02 amplifier to a Kozhevnikov integrator [2], which produced the curve of the pulse amplitude in respiratory volleys. In addition, the pneuograms and electromyograms were recorded on a strip chart oscillograph (type N-105).

Six series of experiments were performed on sixty male rats of the Wistar strain, weighing 280-310 grams. The animals were kept for one hour in air to record the background and then placed for the same period of time in a barochamber with a volume of 80 liters, filled with various gas mixtures under pressure. In all cases, the partial pressure of oxygen was kept at the atmospheric level. The following mixtures were used: nitrogen-oxygen mixture at pressures of 2.5 and 9 kg/cm^2, helium-oxygen mixtures at 9 kg/cm^2 and "elegaz"-oxygen[3] at a pressure of 1 kg/cm^2 and without pressure.

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Results of the Study and Their Evaluation

The experiments which were performed revealed changes in the investigated indices even when the pressure of the nitrogen-oxygen mixture was raised to 2 kg/cm^2 (3 ata). During the time the animals were under pressure, the amplitude of the electrical impulses in the external intercostal muscle and the time of the respiratory cycle increased by 17% ($p < 0.05$). When the pressure of this mixture was raised to 5 and 9 kg/cm^2, the changes in the investigated parameters were more clearly evident. But, at a pressure of 9 kg/cm^2 the amplitude of the oscillations in the electrical activity in the inspiratory muscle during the period spent in the barochamber increased by 27% while the time of the respiratory cycle increased by 12% in comparison to the original level in air (see the Figure, B). In the helium-oxygen mixture at the same pressure (fourth series), the investigated parameters remained practically the same (see the figure, C).

In view of the fact that in a helium-oxygen atmosphere under pressure, when the density of the gas mixture was nearly equal to that of air (at

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[3] Oxygen in sulfurhexachloride (SF_6).

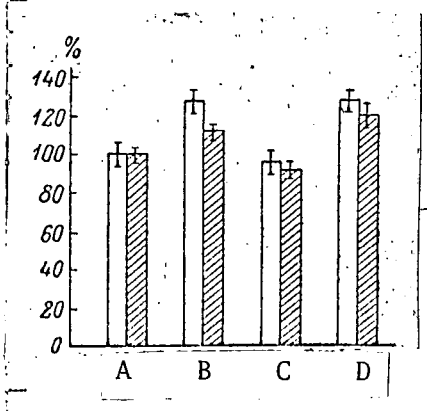
atmospheric pressure), no changes in the investigated parameters were observed, we can conclude that increasing the amplitude of the electrical activity in the inspiratory muscle and increasing the time of the respiratory cycle in the first three series of experiments was caused by an increase in the density of the inspired air.

To check this finding and exclude the possible narcotic effect of nitrogen under pressure, we used sulfurhexachloride, called "elegaz" in the next two series of experiments as the diluent for the oxygen (the density of this mixture was approximately five times higher than the density of air). With the "elegaz"-oxygen medium, we obtained results similar to those in the preceding studies in the nitrogen-oxygen atmosphere under pressure. The amplitude of the impulses of electrical activity in the inspiratory muscle increased by 28%, while the time of the respiratory cycle increased by 20% in comparison to the original level in air (see the figure, D). When the pressure of this mixture was increased by a factor of 2, the changes in the investigated parameters were still more evident. Thus, the amplitude of the electrical impulses increased by 65% ($p \leq 0.05$), while the time of the respiratory cycle increased by 25% ($p \leq 0.05$).

Hence, increasing the density of the inspired gas mixture either by increasing the external pressure or by changing the physical characteristics of the gas used for diluting the oxygen produces an increase in the electrical activity in the respiratory volleys of the external intercostal muscles and the time of the respiratory cycle, i.e., the respiration slows down. These changes are evidently caused by an increase in the work of the respiratory musculature due to inspiration of a dense gas mixture and a rise in the resistance to the air flow in the respiratory pathway. /1810

In order to analyze the relationship of the load on the respiratory musculature to the external pressure, we must consider the components of which the resistance to respiration is composed. As we know, the total resistance to respiration consists of three components: elastic, inertial and inelastic resistances [9]. The first arises as the result of the elasticity of the lungs and chest. The elastic properties of the tissues are independent of the

external pressure, since they are made up of water which is practically incompressible. Hence, the elastic resistance does not change under conditions of excess pressure. The inertial resistance is composed of the inertial resistance of the gas and the inertial resistance of the tissue. Under increased pressure, there is an increase in the mass of the gas passing along the respiratory pathways and naturally there is an increase in the value of the inertial resistance. However, as special studies have revealed, this increase is so small that it may be disregarded [17]. Inelastic resistance is made up of the inelastic resistance of the tissues and the resistance of the respiratory pathways. Under conditions of increased pressure, inelastic resistance of tissues does not change and there is only the significant increase in the second component — the resistance of the respiratory pathways.



Electrical Activity of Muscles and Time of Respiratory Cycle in Animals in Gas Mixtures of Various Densities. A, Initial level in air; B, In nitrogen-oxygen mixture at a pressure of 9 kg/cm^2; C, In a helium-oxygen mixture at a pressure of 9 kg/cm^2; D, In an "elegaz"-oxygen mixture at atmospheric pressure. Empty columns, amplitude of electrical activity of muscle in respiratory volleys (in % of original level); shaded column, time of respiratory cycle (in % of original level).

In view of the fact that the gas flow in the respiratory pathways is essentially laminar in nature, with the exception of places where the bronchial tree branches naturally, where vortex formation occurs and the gas flow becomes partially turbulent. On the basis of these considerations and in view of the fact that the movement of the gas along the respiratory pathways occurs due to the slight pressure differential between the alveolar space and the ambient medium, Rorer [9, 23] derived a formula which characterizes the pressure of the gas flow in the respiratory pathways:

$$\Delta P = \dot{V} \cdot K_1 + \dot{V}^2 \cdot K_2,$$

where ΔP is the pressure differential which ensures air flow; $\dot{V}$ is the volume of velocity of the gas; K_1, coefficient of laminar flow (function of dynamic viscosity

laminar flow (function of dynamic viscosity of the gas); K_2, coefficient of turbulent flow (function of gas density).

Hence, the resistance to respiration which is produced by the laminar portion of the flow is directly proportional to the volume velocity and the dynamic viscosity, while the turbulent portion is directly proportional to the density and the square of the volume velocity of the inspired air.

Evidently Rorer's formula makes it possible to calculate in advance the level of resistance to respiration under conditions of high pressure on the ambient medium, since the gas density at any pressure is known. However, as studies have shown, the equation derived by Rorer corresponds to conditions at atmospheric pressure, but at high pressure it does not agree with the data obtained by experimental means [16]. /1811

The nature of gas flow in tubes, as we know, is determined by the Reynolds number. It has been determined experimentally that if the Reynolds number approaches 2,000, flow will change from laminar to turbulent. Increasing the density and rate of movement of the gas promotes the development of turbulence, while increasing the dynamic viscosity retards this process. In other words, as the external pressure increases and the density of the inspired mixture rises, the turbulence will develop in the gas flow that was previously laminar. If we take the Reynolds number into account, we can determine more precisely the value of the resistance to respiration with increased pressure of the ambient medium [23], although not everyone shares this view [8, 9]. Several investigators have suggested another concept for the characteristic of the air flow in the respiratory pathways — the index of the pressure differential which is directly dependent on the gas density [8].

Hence, the fraction of the turbulent flow during respiration under excess pressure increases and there is a corresponding increase in the dependence of the resistance to respiration and the load on the respiratory musculature on the density of the gas mixture.

This is one of the reasons why it is preferable under conditions of increased pressure to use gases with low density, especially helium [16]. It was found on an "artificial lung" apparatus at atmospheric pressure that the

value of the resistance to the airflow is 4.6 times greater than for helium [1]. Increasing the resistance to respiration leads to activation of the respiratory musculature. Thus, when breathing air at atmospheric pressure under conditions of constriction of the trachea, there is an increase in the bioelectric activity of the diaphragm; however, when the air is replaced by a mixture of helium and oxygen, the work of the diaphragm muscle returns to normal [3]. In our experiment also, using a helium-oxygen mixture under excess pressure, we found that the electrical activity of the external intercostal muscles does not change, but does increase markedly in a dense "elegaz"-oxygen mixture at atmospheric pressure. The slowing of respiration which was observed in the rats at this time is evidently of an accommodative nature, aimed at the energetic optimization of the mode of pulmonary ventilation required under the given conditions. It has been shown that at a pressure of 10 kg/cm^2 and a minute respiratory volume of 10 ℓ/min, the work of respiration in a nitrogen-oxygen mixture in comparison to one of helium and oxygen is increased by approximately 1.45 times, while when the MRV increases the frequency of respiration is also somewhat greater [5].

Data are available which indicate that an increase in the work of the respiratory musculature under conditions of increased atmospheric pressure leads to highly unfavorable consequences — accumulation of carbon dioxide in the alveolar air [10, 12, 20]. Here we can draw a slight analogy with clinical observations. Thus, individuals with obstructive diseases of the respiratory pathways and chronic pulmonary insufficiency also sometimes display an increased concentration of carbon dioxide in the alveolar air [4, 21]. Under conditions of excess pressure, this phenomenon evidently must be viewed as a disruption of the function of the external respiration.

Hence, the significant increase in atmospheric pressure and density of inspired gas leads to the development of electrical activity and a rise in the load on the respiratory musculature, as well as changes in the parameters of the external respiration, and may lead to decompensation of the respiratory function.

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